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VAN DIE REDAKSIE

SIMPATEKTOMIE VIR DRUKVERHOGING

Amper soos modes, is operasies geneig om aan die orde van die dag te wees en daarna in genoeglike onopsigtelikheid na handboeke uit te wyk. Sekere prosedures (soos gastrojejunostomie byvoorbeeld) is teenswoordig net so ongewild soos die lae roksoom. Een hiervan is Smithwick se lumbo-dorsale simpatektomie vir drukverhoging, waarvan 'n vooraanstaande chirurg, C. Wells, onlangs geskryf het: ‚Fewer operations are more dead today'.[1] Min mense sal die waarheid van hierdie stelling betwyfel, want dit skyn of chirurge, nie minder algemeen as interniste nie, universeel wantrouig is om hulle strawwe gevalle van drukverhoging aan die operasie te onderwerp. Dit mag wellig wees dat daar te veel daarvan in die eerste plaas verwag is want, soos Wells aantoon, sal simpatektomie skaars die primêre oorsaak van die essensiële drukverhoging, wat dit ookal later mag blyk te wees, beïnvloed. Maar deur ,'n opdamming in die bloedvate van die ingewande en spiere van die onderste ledemate' te skep, word die perifere weerstand verlaag, en is dit geneig om sodoende die hemodinamika van die liggaam tot normale vlakke te herstel. Alhoewel simpatektomie dus vanuit 'n genesende oogpunt waarskynlik as 'n mislukking bestempel kan word, mag die verligtende hoedanigheid daarvan letterlik lewensreddend wees. Hoofpyne byvoorbeeld, sal verban word—'n ietwat ingrypende voor-operatiewe voorspelling wat deur Wells as gereverdig aangeneem word—en die retinale tekens van die siekte word tot stilstand gebring, indien nie verbeter nie, en dit mag die gesigsvermoë in stand hou.

Die vernaamste beswaar teen simpatektomie is dat die afname in bloeddruk nie standhoudend is nie. Verlenging van Smithwick se torako-lumbale reseksie om die hele borsketting in te sluit, maak weinig verskil. Ervare navorsers by verskeie klinieke het hierdie bevinding bevestig,[2] en mag as gevolg daarvan hierdie prosedure verdoem het, ten spyte van die verligting wat dit mag meebring. Daar mag bespiegelings gemaak word oor die prognostiese maatstawwe wat gebruik behoort te word, ten einde die doeltreffendheid van hierdie prosedure te beoordeel. Hoofpyn is glad te 'n inkonsikwente kenmerk;[3] algemene welsyn en afwesig-

EDITORIAL

SYMPATHECTOMY FOR HYPERTENSION

Operations tend to have their day, rather like fashions, and then regress into comfortable obscurity in the text-books. Certain procedures (such as gastrojejunostomy, for example) are as unpopular today as the low hemline. One of these is Smithwick's lumbo-dorsal sympathectomy for hypertension, of which an eminent Scottish surgeon, C. Wells, recently wrote: 'Fewer operations are more dead today'.[1] Few people will doubt the truth of this statement, for surgeons no less than physicians seem to have become universally shy of submitting their severe hypertensive cases to the operation. It may well be that too much was expected of it in the first place, for, as Wells points out, sympathectomy will scarcely influence the primary cause of the essential hypertension, whatever it may one day be found to be. But by creating a 'splanchnic and lower limb muscular pool' it lowers peripheral resistance and thereby tends to restore the bodily haemodynamics to normal levels. While sympathectomy can thus probably be described as a failure from the curative point of view, its palliative propensities may be literally life-saving. Headaches, for instance, will be banished—a rather sweeping pre-operative prediction that Wells believes to be justifiable—and the retinal signs are halted, if not reversed, and this may maintain the eyesight.

The principal objection to sympathectomy is that the fall in blood pressure is not maintained. Extension of Smithwick's thoraco-lumbar resection to include the whole thoracic chain makes little difference. Experienced workers in many clinics have substantiated this finding,[2] and as a consequence may have condemned the procedure despite the palliation it may bring. One may speculate on the prognostic criteria that should be used to judge the efficacy of the procedure. Headache is far too inconsistant a feature;[3] general well-being and absence of discomfort far too

heid van ongemak by verre te vaag en onderworpe aan uitwendige invloede; terwyl 'n laer bloeddruklesing *nie op sigself* die finale proef vir sukses mag wees nie. Daar bestaan ongetwyfeld 'n ligte graad van ‚bloeddruk' neurose, veral by gevalle van drukverhoging en andere wat dink dat hulle kandidate vir ‚bloeddruk' mag wees.[4] By sulke persone sou na-operatiewe verligting van hoofpyn waarskynlik 'n tydperk van totale simptomatiese verligting inlui, terwyl die herverskyning van drukverhoging (of selfs die vermoede daarvan) ongetwyfeld die simptome, wat voor die operasie aanwesig was, sou vererger. Indien die prognostiese berekening van simpatektomie moeilik is, dan is die keuring van gevalle vir operasie nog moeiliker. ‚Swak risiko' gevalle hoef nie noodwendig uitgesluit te word nie; alhoewel die operatiewe sterftesyfer verminder indien dit gedoen word, word die gunstigste resultate daardeur verbeur.[1] Die alternatiewe weg van mediese behandeling is altyd beskikbaar en soms—soos ook met ander toestande—word die chirurg alleen ingeroep wanneer die internis bereid is om mislukking te erken, en wanneer die moontlike voordeel van simpatektomie nie verder verkrygbaar is nie. Ten spyte van die wesenlike vordering met mediese behandeling van hierdie toestand gedurende die afgelope dekade, bly die feit staan dat daar nog geen duidelike aanduiding vir chirurgiese inmenging tot nog toe te voorskyn gekom het nie. Van pasiënte wie se bloeddruk dringend afgebring moet word, skryf Pickering: ‚Should they be too stupid or uncooperative for this (medical) therapy, or should he or she loathe (as some do) such an exacting regime, sympathectomy should be considered'.[5] Pickering se denkwyse onderskryf tot 'n sekere mate die ietwat proefondervindelike aard van die aanduidings vir die uitvoering van die operasie; billikheidshalwe egter, moet dit vermeld word dat niemand met sekerheid kan sê of dit moontlik van enige nut sal wees nie. Tewens is die enigste werklike konkrete gevolg van herhaalde selektiewe toetse skynbaar die verminderde risiko dat gevalle van geneesbare drukverhoging (bv. eensydige nieraandoening) oor die hoof gesien word.[6] Aangaande mediese behandeling het Pickering persoonlik twyfel uitgespreek—‚all apparently successful forms of therapy have a high nuisance-value'—en 'n ander navorser het heksametonium terdeë verdoem met slegs 'n sweem van lof. Die geneesmiddel is nuttig, skryf hy,[7] as die pasiënt gewillig is om te vergenoeg met ‚blurred vision, dryness of the mouth, constipation (even paralytic ileus), urinary retention, abnormal susceptibility to spirits, embarrassing faintness, or syncope and impotence; and if his doctor is ready to prescribe suitable laxatives, cholinergics, adrenergics and to teach the patient and his family to take and record blood pressure'.[7]

Terwyl simpatektomie definitief in onguns geraak het, het daar nog nie met die tyd enige oortuigende alternatiewe vooruitsig vir die lyer aan drukverhoging gekom nie. Intussen kan ons nie bybring om 'n metode te vergeet wat ten minste die vooruitsig van tydelike vague and liable to extraneous influences; whilst a lower blood-pressure reading may not *in itself* be the final test of success. There is little doubt that a mild 'blood pressure' neurosis does exist, particularly amongst hypertensives and others who feel that they may be candidates for 'blood pressure'.[4] In such persons post-operative relief of headache would probably herald a period of complete symptomatic relief, whilst a return of hypertension (or even the suspicion of it) would doubtless aggravate the symptoms that were present before the operation. If the prognostic assessment of sympathectomy is difficult, the actual selection of cases for operation is even more so. 'Bad risk' cases should not necessarily be excluded; although the operative mortality is lowered if this is done, the best results are then missed.[1] The alternative course of medical treatment is always available and sometimes—as in other conditions as well—the surgeon is only called in when the physician is prepared to admit defeat and when the possible benefit of sympathectomy is no longer attainable. Despite the real advances in the medical treatment in this condition in the last decade, the fact remains that no clear indication for surgical intervention has yet emerged. Of patients whose blood pressure must be lowered urgently, Pickering writes: 'Should they be too stupid or uncooperative for this (medical) therapy, or should he or she loathe (as some do) such an exacting regime, sympathectomy should be considered'.[5] Pickering's attitude tends to underline the somewhat empiric nature of the indications for performing the operation; in all fairness, however, it should be stated that no one can say with certainty whether it is likely to do any good. In fact, the only really concrete thing to come out of the battery of selection tests appears to be the diminishing risk of overlooking cases of curable secondary hypertension, e.g. unilateral renal disease.[6] Of medical treatment, Pickering has himself expressed doubts—'all apparently successful forms of therapy have a high nuisance-value'—and another worker has thoroughly damned hexamethonium with the very faintest of praise. The drug is useful, he writes,[7] if the patient is prepared to put up with 'blurred vision, dryness of the mouth, constipation (even parylytic ileus), urinary retention, abnormal susceptibility to spirits, embarrassing faintness, or syncope and impotence; and if his doctor is ready to prescribe suitable laxatives, cholinergics, adrenergics and to teach the patient and his family to take and record blood pressure'.[7]

While the pendulum of favour has definitely swung away from sympathectomy, time has not yet brought any convincing alternative hope for the hypertensive. Until such a time, one cannot afford to forget a procedure that at least holds the prospect of temporary

verligting inhou vir 'n toestand wat nog steeds 'n uitdaging vir die mediese wetenskap bly nie.

1. Ogilvie, W. H. (1956): J. Roy. Coll. Surg. Edinb., **1**, 123.
2. Chris, S. M. (1956): Brit. Med. J., **1**, 665.
3. Platt, R. en Stanbury, P. S. W. (1950): Lancet, **1**, 651.
4. Ayman, D. *et al.*, aangehaal in 'n redaksionele artikel (1956): *Ibid.*, **2**, 1031.
5. Pickering, G. W. (1955): *High Blood Pressure.* Londen: Churchill.
6. Van die Redaksie (1956): Lancet, **2**, 1031.
7. Palmer, R. S. (1955): New Engl. J. Med., **252**, 22.

relief for a condition that continues to remain a challenge to medicine.

1. Ogilvie, W. H. (1956): J. Roy Coll. Surg. Edinb., **1**, 123.
2. Chris, S. M. (1956): Brit. Med. J., **1**, 665.
3. Platt, R. and Stanbury, P. S. W. (1950): Lancet, **1**, 651.
4. Ayman, D. *et al.*, quoted in Editorial (1956): *Ibid.*, **2**, 1031.
5. Pickering, G. W. (1955): *High Blood Pressure.* London: Churchill.
6. Editorial (1956): Lancet, **2**, 1031.
7. Palmer, R. S. (1955): New Engl. J. Med., **252**, 22.

THE DILEMMA OF MEDICAL EDUCATION

It is a well-known fact, and historical references will confirm it, that outstanding work of vital importance, and more particularly of original character, has usually been produced by men and women before the age of 40. It appears that senescence for original thought comes upon us shortly after we are 30 and, by the time the age of 40 is reached, experience, caution, reserve and family responsibilities have so weighed down the other aspects of our psyche that most of us are unwilling or unable to make original observations or to suggest original lines of thought which may lead to new discoveries.

The growth of every individual branch of science, and of medicine in particular, has been phenomenally fast in the last few decades. As a result, the student has to acquire an immense number of new facts in the basic sciences as well as in clinical subjects. Anybody who has been on the Board of Faculty at a University will notice that, over the years, more and more lectures and more and more subjects are being crammed into the medical student's curriculum. As the academic years become overloaded, additional years are added; our most recent effort in this direction has been to make the medical course a full academic 6 years with a seventh thrown in for good measure as an additional year. So, 7 years after his matriculation, at an average age of 24, the medical man starts his career. At that stage he may go into general practice but, should he wish to specialize, a still further period of preparation lies ahead of him, consisting of a minimum of 5 years. The youthful specialist, therefore, is today youthful in name only. He is a man of over 30, and he starts his active practising life then.

This young man has but 5 or 6 years to make his mark on original medical thought, and the more esoteric and restricted his subject, the longer will he take to get to this stage and the fewer years will remain before the 'dead line'. This then is the problem. How, while giving the young doctor a sufficient grounding in all the subjects, are we going to enable him to have sufficient years left of that vital and precious decade between 30 and 40? We must find a solution, or original scientific thought in medicine may come to a halt.

Perhaps the solution might be found in reducing the number of lectures and the time spent on some of the less vital subjects in the curriculum. The worst judges of the importance of their subjects must inevitably be the professors. It would not be reasonable to ask a man who is immersed in his own specialized subject to reduce the time that is necessary to learn its fundamentals. The more he knows, the more he feels should be known by the aspirant candidate.

Another possible solution may well be the reconsideration of the training requirements for the general practitioner. Twenty-five years ago it was possible to get a diploma which could be registered 4 years after matriculation. The conjoint diploma, like many medical degrees in English and Scottish universities, could be achieved in 5 years. Many of our most distinguished practitioners entered their life's work with no longer training than this, and many of them were the very persons to whom we owe much of the advances that have occurred in medicine; no one will suggest that the holders of these diplomas or degrees are any the worse practitioners of their art or that they have not been capable of assimilating facts as advances have taken place.

Have we bitten off more than we can chew? Have we not tried to make the average doctor into a specialist? Cannot means be found to reduce the time required to qualify, or must we allow the process to take its course and go on increasing our curriculum? Carried to its logical conclusion, this will mean that ultimately a man will qualify for practice too late to be able to give the best service even as a specialist. Where are we to stop? The problem requires careful thought. It is becoming urgent.

UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 6 days ended 12 December 1956.

Plague, Smallpox: Nil.

Typhus Fever: No further cases have been reported from the Cradock Municipal area and the districts of Queenstown and Fort Beaufort since the notification of 15 November 1956. These areas may now be regarded as free from infection.

Epidemic Diseases in Other Countries:

Plague: Nil.

Cholera in Chittagong, Dacca (Pakistan).

Smallpox in Ahmedabad, Allahabad, Bombay, Calcutta, Delhi Jodhpur, Madras, Visakhapatnam (India); Baghdad, Basra, Margil, Mosul (Iraq); Karachi (Pakistan); Nairobi (Kenya).

Typhus Fever in Baghdad (Iraq); Alexandria (Egypt).

DIABETES IN PREGNANCY*

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A good general working rule is that, should a surgical or medical ailment be detected in a pregnant patient, treatment, with few exceptions, is directed at the disease and the pregnancy is left undisturbed. Thus, should a pregnant woman develop an acute intestinal obstruction, acute appendicitis, pneumonia or typhoid, these conditions are treated regardless of the pregnancy. The onset of pregnancy in a patient suffering from a chronic illness, e.g. heart disease or pulmonary tuberculosis, throws an added risk on the patient. The complications confronting her are known. She is therefore steered clear of these in pregnancy, labour and the puerperium with all the skill available. In most cases the cooperation of the patient is of prime prognostic significance. However, should the patient fall into one or more of these pitfalls that dot her way, the accoucheur, wisely working in conjunction with a physician, will have all in readiness. The underlying principle in these cases is that it usually is safer both for the mother and the foetus to leave the pregnancy to run its full course.

This principle received a set-back by the introduction of the newer methods of treatment of certain endocrinal dysfunctions. Keynes[6] has shown that a pregnant patient suffering from hyperthyroidism runs the risk of giving birth to a cretin with an enlarged thyroid gland if she is treated with thio-uracil. Should a woman suffer from diabetes mellitus, her chances of becoming pregnant, were negligible before Banting and Best's dramatic discovery of insulin in 1921.[1] Her chance of survival, should she have become pregnant before that era, was roughly 50%, as were those of her unborn babe. It is of interest to note that not only has this discovery made life worth while to innumerable sufferers but the maternal death-rate from diabetes has been reduced to less than 1%; the foetal loss, however, still remains in regions varying from 10 to 50%. This phenomenon has led to much interesting work and speculation. It does signify that diabetes does not permit us to conform to the rule of treating the medical complaint and leaving the pregnancy alone. Diabetes does affect pregnancy and is affected by it. Of great interest also is the fact that not only does the diabetic react in her peculiar way to pregnancy but the woman who will develop diabetes later, i.e. the pre-diabetic, may behave in a similar way. All these features have been uncovered since Reveno[12] treated the first pregnant diabetic in 1923 in the USA. The baby died neonatally. It was left to George Graham[5] in England in 1924 to report the first diabetic to be treated with insulin who was delivered of a live, normal baby. Kriss and Futcher[7] in 1948 stated that 77·1% of all mothers who delivered babies of 10 lb. and heavier subsequently develop diabetes.

Before the advent of insulin therapy, diabetics were usually infertile. The genital glands and organs in untreated cases remain relatively quiescent. One of our patients gave the following story demonstrating the fact that diabetes may be responsible for the failure of complete development of puberty. Adequate therapy soon had her genitals functioning normally.

A 24-year old patient stated that she was in poor health and had not menstruated until 17½ years old. Medical examination at that time disclosed diabetes. Dietary corrections and insulin were prescribed, and almost immediately she regained her health and started menstruating regularly. Her periods remained regular and she became pregnant shortly after marriage.

The following case is of interest for her story shows that uncontrolled diabetes will depress genital function, which is completely restored once adequate therapy is instituted.

43-years-old Mrs. J. W. stated that menstruation had been normal in every way. During the first 10 years of married life she gave birth to 5 normal, live babies at term. All the babies weighed over 10 lb. In 1938 her 6th pregnancy terminated spontaneously in the delivery of a stillborn 14-lb. baby. There was no story of glycosuria, post-partum haemorrhage or shock. From that time onwards her periods were scanty and irregular. Periods of amenorrhoea lasted up to 4 months. Despite normal married life she did not become pregnant. In December 1946 she started complaining of polyuria and polydipsia. Two months later she consulted a doctor, who confirmed the diagnosis of diabetes. Insulin therapy was immediately instituted. In July 1947, i.e. following upon 9 years of secondary sterility, and within 5 months of the institution of insulin therapy, she was found to be 2 months pregnant. She gave birth to a macerated foetus weighing 12 lb. 2 oz. She continued with insulin therapy and became pregnant once more. After careful supervision a Caesarean section was done during the 37th week of pregnancy. The live female baby weighed 8 lb. 3 oz.

It has been learnt since the advent of insulin therapy that pregnancy, labour and the puerperium have an adverse effect on the maternal diabetes. Diabetes in its turn may have an effect on these functions, but in addition, it has a decided effect upon the foetus.

EFFECT OF PREGNANCY, LABOUR AND PUERPERIUM ON DIABETES

Effect of Pregnancy. During the *early months of pregnancy* many women are anorexic and suffer from nausea and vomiting. It is well known that diabetics

* From a lecture delivered at a meeting of the Cape Western Branch of the Medical Association of South Africa on 28 September 1956.

require a well controlled diet and possibly a finely controlled insulin-administration. This balance is obviously upset by not taking a sufficient quantity of the right food or by vomiting some of it. Throughout the early months of pregnancy, therefore, a careful urinalysis is essential. From about the *4th or the 5th month* onwards the renal threshold drops. The urinary sugar estimation becomes a fallacious guide. The drop in the renal threshold results in the passage of large quantities of

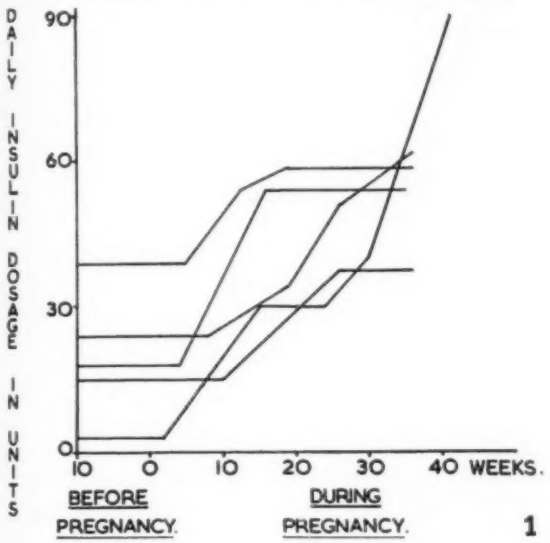

Fig. 1

sugar. This in turn upsets the metabolic balance. The presence of lactosuria is an added difficulty. Treatment therefore becomes difficult as urinary examinations are wrongly interpreted. In these cases frequent blood-sugar estimations should be done and treatment be instituted accordingly. During the *2nd and 3rd* trimesters the insulin requirement in at least 75% of cases rises—yet a further difficulty in the assessment of the patient. Because of these imbalances the patient develops ketosis rather readily during the latter months of pregnancy. Fig. 1 demonstrates the increased insulin requirements rather well. These were taken as examples from a few of the patients.

It is thought that the reason for this deterioration of carbohydrate tolerance may well be overactivity on the part of the pituitary during pregnancy i.e. its diabetogenic factor amongst others. The evidence is abundant that the diabetes becomes worse during pregnancy and the patient suffering from this disease therefore requires careful medical handling. Also, a latent diabetic is very often shown up by pregnancy.

Effect of Labour. Muscular activity requires energy. Carbohydrate is thus rapidly utilized for this purpose in labour. This results in hypoglycaemia. The insulin requirement may suddenly drop of its own accord just before or during the onset of labour. The insulin/carbohydrate requirements therefore necessitate very careful balancing. In prolonged labour diabetic control becomes a major problem, because of the difficulty of being unable to regulate food intake properly and the impairment of normal digestion.

Effect of Puerperium. Immediately after delivery there is an improvement in the carbohydrate tolerance. Should the patient's diabetes have deteriorated even to

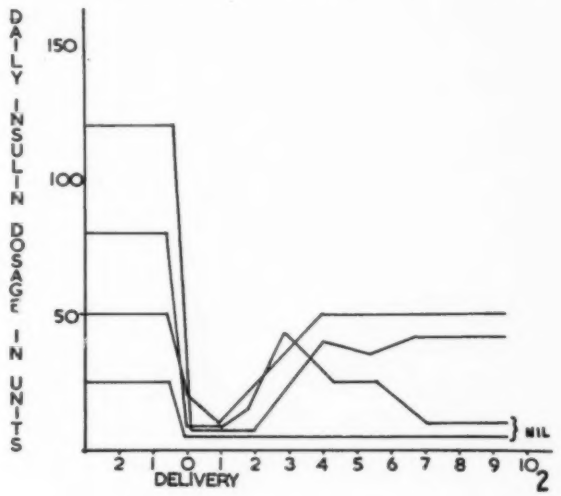

Fig. 2

the point of diabetic retinitis during pregnancy, this condition improves after delivery.[9] The improvement may be a reversal to the patient's pre-pregnant condition or there may even be a permanent betterment. Fig. 2 shows the marked drop in insulin requirements immediately after delivery.

From the above it will be seen that pregnancy has a temporary deleterious effect on diabetes. There are many pitfalls along the way. The unwary may drop his patient into any one of these pitfalls. Once delivered, the old balance is soon regained or in some cases a better one is established.

EFFECT OF DIABETES ON PREGNANCY AND LABOUR.

Many incompletely verified statements have been made about the effect of diabetes upon pregnancy and labour. It has been stated that diabetes predisposes to abortion. This was not found in this series. Out of 127 diabetics treated in the University of Cape Town unit during the period 1 January 1952 to 30 June 1956, only 2 patients aborted spontaneously. In another case, pregnancy was terminated at 3 months because of severe hypertension and albuminuria. This might not be a true reflection, for patients may well abort without being diabetic. On the face of the facts presented, abortion cannot be accepted as being due to controlled diabetes. There seems to be a higher incidence of hypertension and albuminuria with diabetes. Hydramnios, unless marked, is a difficult entity to establish. It is stated that the incidence of hydramnios is high in diabetics.[11] Prema-

ture labour has been found to be frequent in diabetics.[2] Pregnancy was terminated in our antenatal cases and therefore no authoritative statement can be made. It seems that many diabetics do not respond well to induction of labour. It is also often stated that they are prone to inferior labours. These are matters that are difficult to assess and the answer will come from those who study large numbers thoroughly. That a large baby may give rise to complications of labour, especially impaction of the shoulders, etc. is well known and requires no further exposition. It is stated that the placenta ages rapidly in diabetics.[13] This is given as one of the reasons for the high intra-uterine foetal death-rate. As stated above, diet and insulin therapy has reduced the maternal mortality rate to less than 1%. In this series not one mother was lost. The foetal mortality rate is not very markedly decreased by maternal insulin-therapy. This fact has led to much theorizing and active work. What is known is that, should the pregnant mother's diabetes be treated, there is a high incidence of intra-uterine death. Peculiarly enough, the babies die during the last few weeks of intra-uterine life. The babies are larger than normal. The increase in size is both an increase in weight due to fat and oedema, and an increase in length. The neonatal death-rate is also higher. There is said to be an increased incidence of foetal abnormalities.

These babies, therefore, are born with an inherently poor vitality. They stand labour and the early days of life badly. Although they are large, they should be treated as prematures; otherwise their chances of survival are markedly decreased. This odd behaviour on the part of the foetus within a diabetic woman requires explanation. In 1941, Young[14] published his work done on puppies. The pups were injected daily with doses of anterior-pituitary extract greatly in excess of the amount required for the production of diabetes in the adult dog. The pups responded with increased growth-rate. Those developing diabetes did so very late, i.e. when no further growth would ordinarily be considered possible. Young therefore concluded that the growth hormone and the diabetogenic factor were very closely related. Barns injected anterior-pituitary extract into pregnant rats and produced 100% intra-uterine deaths. These two experiments are of great interest. It seems reasonable to assume that the anterior pituitary gland plays a part in the production of abnormally large babies and it may well be responsible for intra-uterine foetal death. Added to this assumption is the one previously mentioned, viz. that the deterioration of the carbohydrate tolerance is thought to be due to over-activity on the part of the pituitary during pregnancy. As has been pointed out, this deterioration regresses after delivery. All the factors tend to point towards the pituitary. Priscilla White's administration of large doses of stilboestrol and progesterone throughout pregnancy[13] may be beneficial because of the alleged inhibitory action of these drugs on the anterior pituitary gland. By hormonal administration and by terminating pregnancy at 36 weeks she states that 90% of the babies in controlled diabetics survive.

Duraiswami[4] reported that by injecting insulin into developing chicks' eggs, congenital deformities were produced. The deformities were largely prevented by injecting nicotinamide and riboflavin together with the insulin. This experiment might well have a bearing on the high reported incidence of foetal abnormalities reported in the pregnancies of diabetics.

TREATMENT

The management of a pregnant diabetic may be outlined as follows:

The diabetes must be controlled during pregnancy, labour and the puerperium. The pitfalls have been outlined. They must be constantly borne in mind and adequately dealt with should they arise. According to Oakley[9] a poor control raises the chances of foetal loss. He also points out that there is no relation between foetal loss and the time of onset of the diabetes before the pregnancy took place or to the age of onset of the diabetes in the mother. It is in the interest of the mother to be admitted to hospital for proper control from the 32nd week onwards—or earlier still should the disease be difficult or impossible to control. In hospital the control can be established with adequate blood-sugar estimations. Although White's argument seems convincing, it may well be that with the excellent care she gives to her patients she might achieve similar results without hormonal therapy. The results of the M.R.C. experiment in the UK demonstrate that the foetal loss with or without hormonal therapy is practically identical. The administration of vitamins may have a beneficial effect. As the last few weeks are the ones claiming the life of the foetus, and because we do not know why the baby dies, it seems a reasonable argument to remove the baby from the noxious element. It therefore seems a most reasonable procedure to interrupt pregnancy soon after the 36th week. Should the patient not respond to the induction of labour, or should she go into a poor or a prolonged labour, Caesarean section offers her and her baby, with its lowered inherent vitality, the best chances of survival (see Table II). It is imperative to treat the usually large infant as a premature baby. Failure to do so will result in the high neonatal death-rate reported by so many workers. The metabolism of the baby born of a diabetic is exactly the same as that of any premature infant. It is therefore unnecessary to administer glucose to these infants in the large amounts previously thought necessary. However, the development of hyaline-membrane disease seems to be an increased risk. A good oxygen-supply and humid atmosphere are essential. Gastric aspiration immediately after delivery may be an added precaution.

RESULTS

In attempting to follow these lines of treatment and in trying to localize our diabetic gravidae, the results shown in Table I have been attained. It is of interest to note that out of a total of 90 booked cases 7 babies were stillborn and 7 died neonatally; 76 remained alive. The foetal loss, therefore, was 15·5%. Of the foetuses of non-booked mothers 41·2% were stillborn. During the 4½ years surveyed, 124 pregnancies went beyond the 28th week. The foetal mortality rate was 22·6%.

TABLE I. DIABETES IN PREGNANCY. UNIVERSITY OF CAPE TOWN TEACHING HOSPITALS: 1/1/1952–30/6/56

Booked

Year	*Patients*	*LB*	*SB*	*NND*	*Foetal Loss*
1952	9	7	1	1	22%
1953	18	18	0	0	0%
1954	28	20	3	5	28·5%
1955	22	21	1	0	4·7%
1956	13	10	2	1	23%
Total	90	76	7	7	15·5%

Non-Booked

Year	*Patients*	*LB*	*SB*	*NND*	*Foetal Loss*
1952	4	2	2	0	50%
1953	8	2	6	0	75%
1954	18	15	3	0	16·6%
1955	0	0	0	0	0
1956	4	1	3	0	75%
Total	34	20	14	0	41·2%
Grand Total B & NB	124	96	21	7	22·6%

LB=live birth; SB=stillbirth; NND=neonatal death; B=booked; NB=non-booked.

It is a cold comfort to compare this figure with that of King's College Hospital (28%),[3] the Medical Research Council's report on 9 institutions (25%),[8] and Pedersen and Brandstrup's 15%.[10] There is no doubt that more of our babies would have been saved had there been strict observation of the present-day rule that pregnancy should be terminated at the 36th or 37th week, and had all patients attended antenatally.

TABLE II. MORTALITY FROM CAESAREAN SECTION AND VAGINAL DELIVERY

Method of Delivery	*No. of Cases*	*LB*	*SB*	*NND*	*Foetal Loss*
Caesarean Section					
Booked	37	34	0	3	8·1%
Non-Booked	14	14	0	0	0%
Total	51	48	0	3	5·9%
Vaginal Delivery					
Booked	53	42	7	4	20·7%
Non-Booked	20	6	14	0	50%
Total	73	48	21	4	34·3%
Grand Total	124	96	21	7	22·6%

Table II demonstrates that Caesarean section in these patients carries with it a comparatively low foetal loss. The high mortality in the series delivered *per vaginam* is due to the fact that 11 out of the 21 had died *in utero* before the onset of labour. No mother received hormone therapy and no maternal life was lost. It is of interest to note that the findings of the M.R.C. experiment were that hormone therapy had no effect on the foetal survival rate. Pedersen and Brandstrup[10] state that they agree with Peel and Oakley[11] that the severity and duration of the diabetes does not seem to affect the foetal mortality rate. They state that the duration of adequate control during pregnancy is of vital importance to the foetus. It is abundantly clear that the answer to this problem of lowering the foetal mortality rate is as yet unknown. Further work in this field is essential. The methods employed today may well be outmoded shortly, depending upon new discoveries. The *British Medical Journal* of 25 August 1956 contains no fewer than 7 articles and a leader on the possibilities of a new drug. There is yet much to learn. It does seem odd that the effects of the maternal disease upon the infant cannot be prevented—and that the babe must be 'untimely ripp'd from the womb'. The substances affecting the foetus obviously pass through the placental barrier, for the metabolism of the whole organism is affected.

As the complete story of diabetes, including the effects of the disease and its medication, upon pregnancy and the unborn child, unfolds itself, the lives of yet more babies will be saved. White's figure of a 90% survival rate should be surpassed.

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NICHOLS FELLOWSHIP

The Council of the Royal Society of Medicine invites applications for a grant of £225 per annum in aid of research to be carried out to advance knowledge in obstetrics and gynaecology, which will be awarded on the recommendation of the Council of the Section of Obstetrics and Gynaecology of the Society. The place at which the work is to be carried out and an outline of the proposed research must be stated in the application. A preliminary report on the progress of the research must be submitted at the expiration of the first 6 months. The Fellowship will be awarded in the first place for a period of 1 year and, at the discretion of the Council, may be extended for a second year. Applications must be received by the Secretary, Royal Society of Medicine, 1 Wimpole Street, London, W. 1, by 13 May 1957 and candidates must state their position with regard to call-up for Military Service.

FORMULERING VAN 'N BELEID BY DIE BENADERING TOT KARSINOOM VAN DIE BRONCHUS

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J. M. VAN NIEKERK
J. K. LUNDIE
Universiteit van Pretoria

Dit is nie ons plan om op besonderhede in te gaan nie. Almal dra kennis van die algemene kliniese, patologiese en radiologiese aspekte. Almal weet dat die oorsaak onbekend is, maar sterk suspisie bestaan dat straf-rook 'n faktor is. Die siekte kom in groot sowel as klein bronchi voor, en die ligging bepaal grootliks die radiologiese beeld. Die alveolêre tipe, asook die tipe wat in verskrommelde longweefsel ontstaan voorheen die setel van onsteking of besering d.w.s. in littekens, is seldsaam.

1. DOELWIT

Ons doelwit is nie om besonderhede te verstrek nie, maar in wye trekke 'n oorsig van die benadering tot diagnose, veral vroeë diagnose te gee, en om te pleit vir 'n spesifieke beleidsbepaling ten opsigte van hierdie ernstige siekte; 'n siekte wat volgens statistieke aan die vermeerder is.

Zurich Instituut van Patologie skat persentasie vermeerdering van 1906 tot 1945 op 9·7% tot 19·4%.

2. KARSINOOM—TUBERKULOSE—PNEUMONIE—ENS.

Dáár is onvoldoende bewys dat tuberkulose, pneumokoniose ens., die voorlopers van karsinoom is, hoewel dit soms gebeur dat karsinoom by 'n lyer aan een van hierdie siektes ontstaan. Sommige deskundiges skryf dit toe aan die feit dat tuberkulose tans byvoorbeeld meer dikwels by 'n ouer groep aangetref word, d.w.s. wanneer die persoon in die regte leeftyd vir kanker is.

3. PATOLOGIE EN RADIOLOGIE

Teneinde 'n beleid te formuleer, is dit nodig om in kort die patologiese agtergrond van die radiologiese voorkoms te beskryf, asook vir 'n paar oomblikke by die differensiële diagnose stil te staan. Wanneer die letsel in 'n groot bronchus ontstaan, veroorsaak dit die sogenaamde sentrale tipe (Fig. 4). Wanneer dit in 'n kleiner bronchus ontwikkel, ontstaan die perifêre tipe en selfs die superior sulcus- of Pancoast-tipe. Oor die alveolêre tipe gaan ons nie veel sê nie; dit is in elk geval seldsaam.

Sentrale Tipe

Daar ontstaan die endobronchiale tumor wat aanvanklik nie op gewone X-strale sigbaar is nie, hoewel dit simptome kan gee. Die vroeë tipe wat nie as 'n massa sigbaar is nie, kan soms radiologies op plate in inspirasie en ekspirasie gedemonstreer word; laasgenoemde wys dan lokale obstruktiewe emfiseem.[1] Bronchografie en tomografie mag behulpsaam wees en in 'n hoë persentasie van gevalle is bronchoskopie van hulp (Fig. 9).

Die verspreiding vind in die lumen sowel as in die wand plaas en obstruktiewe emfiseem volg. Die kliere in die omtrek word vinnig aangetas en die druk daarvan op die bronchus dra verder by tot die ontwikkeling van atelektase (Figs. 1, 2 en 5). Op hierdie stadium verwag die geneesheer dus indirekte sowel as direkte aanduiding van abnormaliteit op die X-straal foto. Met verdere infiltrasie en die afsny van die bloedtoevoer in die massa ontwikkel daar somtyds holtes (Fig. 6). 'n Karsinomateuse holte is gewoonlik enkelvoudig in teenstelling met gewone longabses wat dikwels meer as een area van nekrose bevat.

Uiteindelik ontstaan daar maligne pleuritis, hoewel primêre long maligniteit kan voordoen as pleuritis. Been-erosie vind veral plaas waar die tumor in die superior sulcus geleë is en in enkele gevalle is daar aantasting van die nervus phrenicus met verlamming van die diafragma, veral aan die linkerkant (Fig. 9). Op 'n laat stadium word dikwels 'n kombinasie van effusie en kollaps gevind; die hart word nie na die teenoorgestelde kant gedruk deur die effusie nie, weens die onderliggende kollaps. Die tragea mag selfs na die aangetaste kant leun.

Hieruit is dit duidelik dat ander moontlike oorsake van die volgende afwykings by die differensiële diagnose oorweeg moet word: Emfiseem, hilus-skaduwee of mediastinale vergroting, atelektase, holteformasie (abses), pleuritis, eensydige verduistering, ens.

Perifêre Tipe

Hiér ontwikkel die tumor in 'n kleiner bronchus en die skaduwee wat ontstaan, is dus perifeer geleë. Die gevolg is die aanwesigheid van 'n ronde, goed-omlynde skaduwee in die longgebied.

Die bronchus wat by voorkeur aangetas word, is dié van die regter bo-kwab, maar karsinoom tas ook al die ander bronchi aan, selfs dié van die regter middelkwab.[2] Die ronde skaduwee is dikwels omring deur onreëlmatige opasiteite, as gevolg van perivaskulêre en peribronchiale infiltrasie (Figs. 7 en 8).

'n Klein induiking of umbilikasie van die ronde massa word as 'n teken van maligniteit beskou.[3] Verkalking is uiters seldsaam.

By die differensiële diagnose moet ander oorsake van 'n ronde skaduwee oorweeg word:

Aneurisme, pneumonitis met of sonder abses-vorming, tuberkuloom, echinococcus kiste, granuloom, benigne tumor soos hematoom, neurofibroom, uitsaaiings, gekapselde effusie, ens.

As daar groot pulmonêre vate met die tumor verbind is, is dit ten gunste van 'n pulmonêre arterie aneurisme. Kurviliniêre verkalking in die rand van die tumor is sterk ten gunste van 'n aneurisme. Sover ons bekend is, is hierdie tipe verkalking nog nooit in 'n karsinoom beskryf nie. Daar is enkele gevalle van hierdie tipe

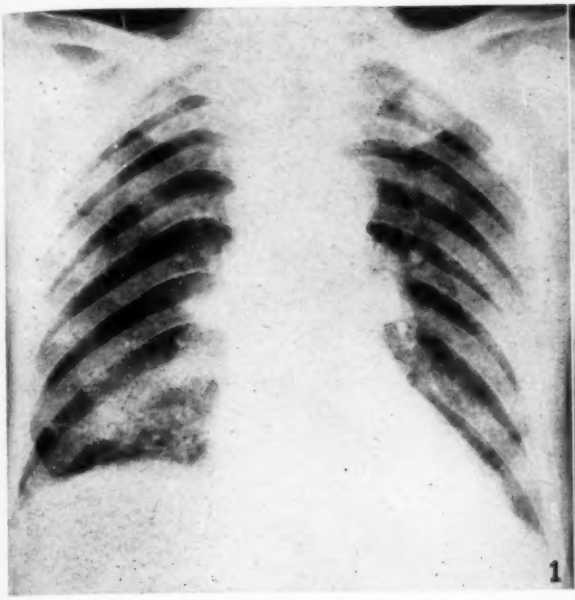

Fig. 1. Karsinoom van bronchus met kollaps van regter middelkwab. Ook uitgesproke verkalking van hiluskliere aan die linkerkant en metastases in die linker negende rib.

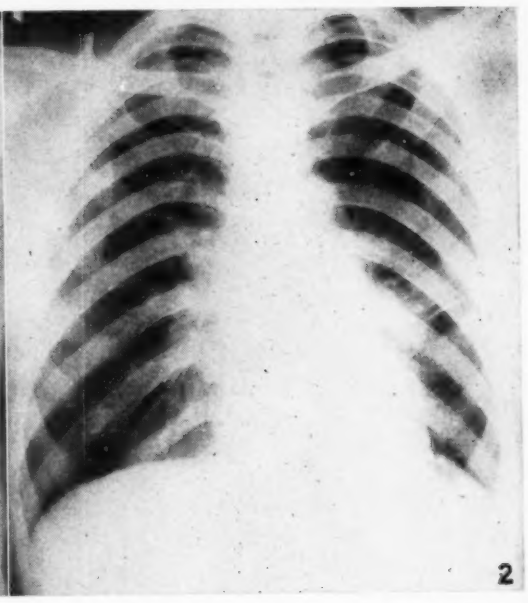

Fig. 2. Karsinoom bronchus met vroeë kollaps lingula segment van linker bokwab.

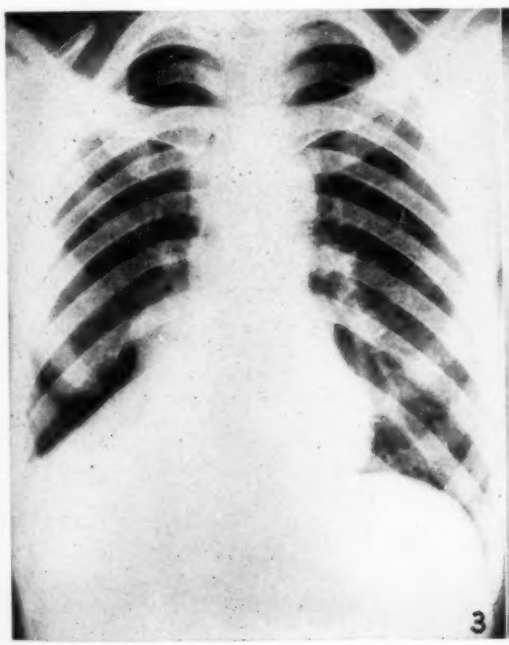

Fig. 3. Karsinoom bronchus met kollaps van regter middel- en onderkwabbe.

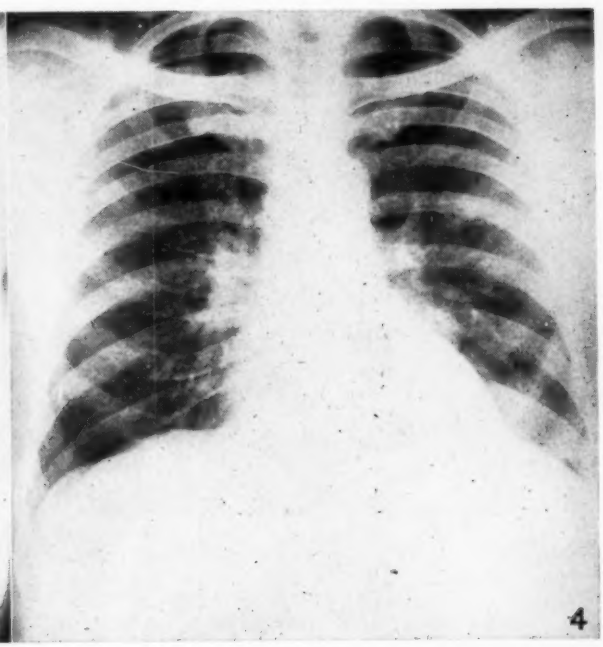

Fig. 4. Karsinoom linker onderkwab bronchus in man van 44 jaar. Ronde massa op linker hartgrens onderkant pulmonêre arterie.

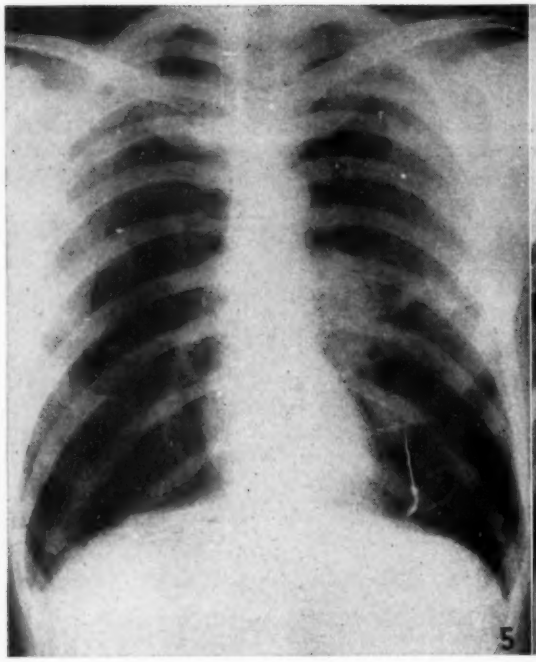

Fig. 5. Karsinoom linker bokwab met groot linker hilus en kollaps van lingula segment.

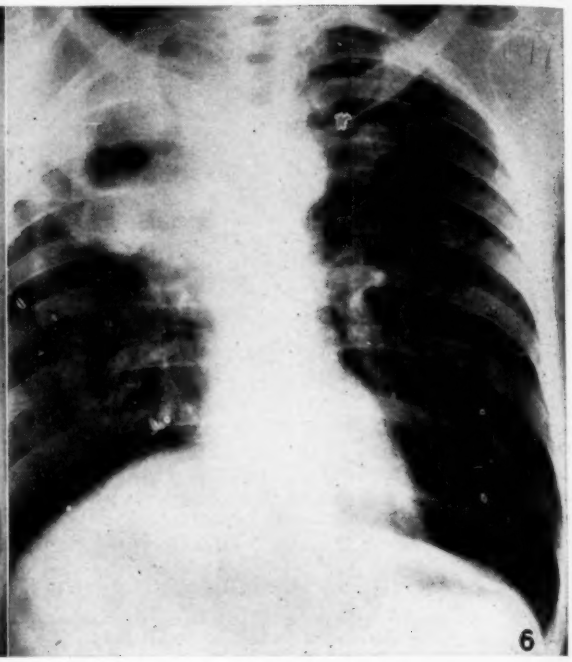

Fig. 6. Karsinoom regter bokwab bronchus met holte formasie; man van 46 jaar.

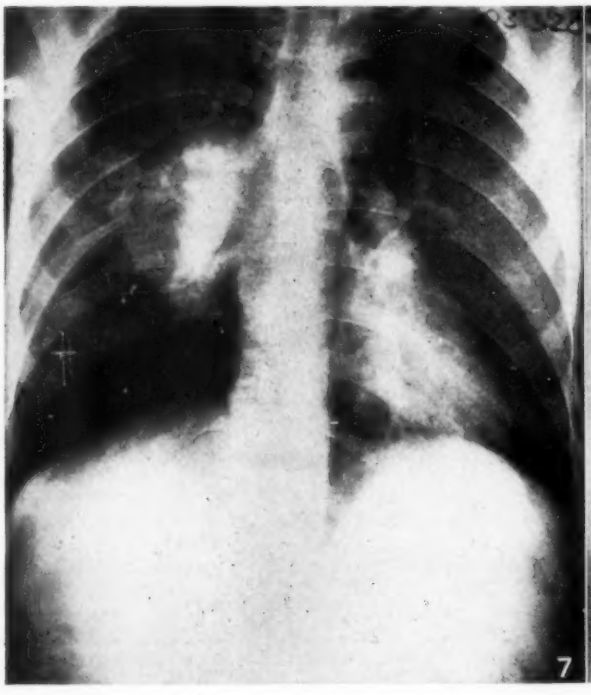

Fig. 7. Posterior-geleë perifêre karsinoom met erosie van werwels. Normale bronchiale boom sigbaar deur massa.

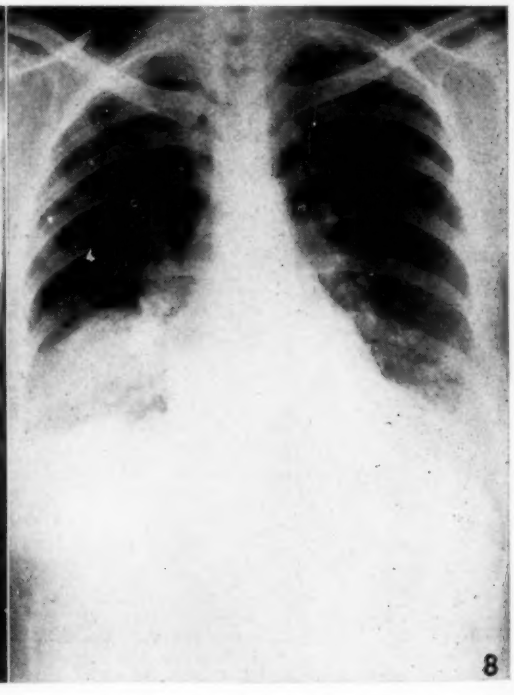

Fig. 8. Posterior-geleë perifêre karsinoom aan die regterkant wat 'n gekapselde effusie naboots.

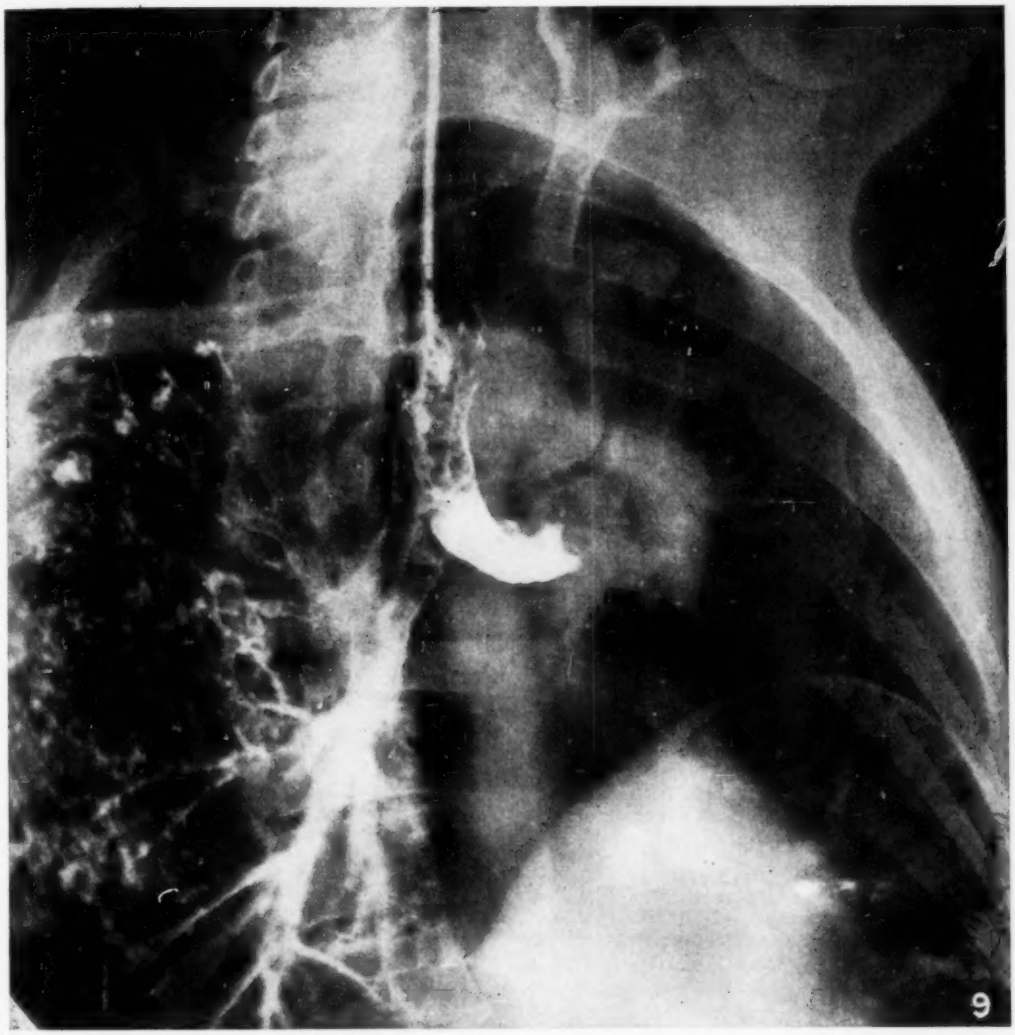

Fig. 9. Bronchogram toon afsluiting van linker hoofbronchus deur 'n karsinoom, met hoogstand van linker diafragma a.g.v. freniese paralise.

verkalking in dermoïde kiste, echinococcus kiste[4] en selfs in retrosternale krop beskryf.[5]

Verandering in vorm met diep asemhaling is ten gunste van 'n kisteuse letsel.

Dit is bekend dat tuberkulomata 'n voorkeur toon vir sekere longstreke, naamlik die apikale segmente van die bokwabbe en apikale segmente van die onderkwabbe.[6]

Pancoast of Superior Sulcus Tumor

Dit is feitlik altyd 'n karsinoom maar in baie uitsonderlike gevalle mag dit benigne van aard wees. Beenveranderinge vind vroeg plaas en die beeld is wel bekend. Dit moet beklemtoon word dat in sommige gevalle die swaartepunt op die skaduwee rus, en in ander gevalle weer op die been-erosie.

Dit moet ook besef word dat hoewel aanvanklik die beeld van perifêre tumore verskillend is van sentrale tipes, uiteindelik as gevolg van infiltrasie, atelektase en pleuritis, daar weinig of geen verskil bestaan nie, sovêr dit die radiologiese voorkooms betref.

4. METODES

Roetine radiologiese ondersoek gepaard met reeds genoemde spesiale ondersoeke is in meeste gevalle nodig om tot 'n diagnose te kom. Dit moet egter beklemtoon word dat die kliniese geskiedenis, die ondersoek van sputum vir maligne selle, bronchoskopie en selfs torakotomie nodig is. Volgens Kerley[7] is angiografie baie waardevol, daar aktiewe mitose van selle altyd liberale bloedtoevoer veroorsaak wat die voorkoms beïnvloed.

Bakteriologiese ondersoek sowel as histologiese ondersoek van tumormassa word gedoen om die bevindinge te korreleer.

Goed-opgeleide persone wat in 'n span kan werk, is natuurlik 'n belangrike vereiste.

5. BELEIDSBEPALING

Nou kom ons by die eintlike punt, d.w.s. die bepaling van 'n beleid.

Die eerste uitgangspunt is vroeë diagnose. Sonder dit, is die saak in elk geval verlore. Karsinoom van die long bied alleen kans tot herstel waar die diagnose vroeg gemaak word. Ons hoef ons dan nie te bekommer oor wat volg nie, want daar bestaan reeds goeie spanwerk tussen borschirurge en radiotherapeute in wie se hande ons die pasiënt oorlaat.

Radiologie is werklik van groot belang, want in 'n groot persentasie van gevalle met of sonder simptome is daar iets verdags te vinde op die roetine of spesiale ondersoeke.

Bronchoskopie is van groot waarde waar die tumor in sekere posisies lê, maar almal is bewus daarvan dat die tumor buite bereik van die instrument mag wees en dus nie sigbaar nie.

Cytologiese ondersoek van bronchiale spoelings of pleurale vog is by 'n groot persentasie van gevalle positief vir maligne selle.

Waar enige twyfel bestaan, is torakotomie die aangewese metode. 'n Korrekte diagnose selfs in die beste hande word nie in meer as 70% van gevalle gemaak nie. Die veiligste metode is dus operatiewe ingreep.

Ons wil voorstel dat enige enkele ronde skaduwee in die long, torakotomie regverdig; selfs in gevalle van enkelvoudige uitsaaiings van 'n hipernefroom of 'n chorion epitelioom, kan genesing na verwydering van die uitsaaiing en die primêre letsel verwag word.

6. WAT OM TE DOEN?

Wat staan ons te doen?

1. Opvoeding van publiek om borssimptome nie te verwaarloos nie.

2. Ons as dokters moet ophou om simptome te behandel sonder om te weet wat die simptome veroorsaak.

3. Ons moet verder gebruik maak van spanwerk, van alle diagnostiese metodes en elke verdagte geval van karsinoom as 'n mediese spoedgeval bejeën. N.B.—Stadige metodes soos opvolg, huidtoetse ens., het geen plek in ons beleid nie.

4. Massa X-straal ondersoek word deur sommige outoriteite voorgestaan hoewel ons nie meer as 10 per 10,000 verwag nie, en hoewel in verkeerde hande moontlike lyers aan intensiewe psigologiese en ekonomiese trauma blootgestel word.

Ons hoop lê daarin dat deur navorsing die een of ander laboratorium of radio-aktiewe isotope-toets uitgewerk kan word om die diagnose te maak.

'n Radio-terapeutiese toets met groot dosisse is soms van hulp. In afwesigheid daarvan is daar niks meer as om te doen wat reeds gesê is nie, n.l. om alle verdagte gevalle die beste radiologiese en ander ondersoek te laat ondergaan en indien daar nog onsekerheid bestaan, torakotomie aan te beveel.

Die beeld wat ons geskilder het, is nie 'n rooskleurige een nie, maar benadruk die noodsaaklikheid van spanwerk om 'n vroeë diagnose te bewerkstellig.

OPSOMMING

Die algemeenste radiologiese bevindinge by karsinoom van 'n bronchus is aangehaal, en die belangrikheid van spanwerk om 'n vroeë diagnose te bewerkstellig, word benadruk.

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QUESTIONS ANSWERED : ANTWOORDE OP VRAE

Q. A lady has recently consulted me. Her first child aged 13 is a healthy girl. Her second child aged 11 is a mongoloid idiot. She is anxious to have more children but is afraid to do so. There is no family history of mongolism or of mental disorder. What am I to advise her?

A Physician replies

The incidence of mongolism rises enormously with the age of the mother. Thus a mother over 45 has a 2% chance of a child being mongoloid, while only 1 in some 600 pregnancies in the general population has this unfortunate outcome. There is probably an increased likelihood that, once a mongoloid has been produced, a further child will be similarly affected, but this chance is only slightly greater than the normal mathematical expectation. Certainly one can never *promise* a favourable outcome, but one's advice can only be to go ahead!

A Paediatrician replies

The questioner does not give sufficient information on which to base an informed opinion. The details which would help are (1) the lady's age, (2) the number of her pregnancies, (3) the history of the pregnancy resulting in the second child with regard to haemorrhage, infection, or X-rays in the early months, etc.

Presumably the lady is over 33 years, and had the second child when in her early twenties which would suggest an extrinsic cause. If that is so she would run no more than the normal risk of any woman over 35 years of having an abnormal baby and should be advised to fulfil her ambition. The sooner she does so the better since there is a small but definite increase in the risk as the maternal age increases. If she has had a number of abortions or has any gynaecological abnormality she should have special advice from that angle.

She cannot be given a guarantee of success but the risk in her case would appear to be small and the phychological effect of a successful pregnancy would be far-reaching, not only as regards both parents but also as it might easily concern the matrimonial prospects of the 13-year-old daughter. The answer would appear to be, from most aspects, favourable. Even if none of the above aetiological factors can be invoked she has a 97% chance of having a normal baby, or several of them.

AORTOGRAPHY: THE SAFEST MEANS OF PLACENTAL LOCALIZATION?

A PRELIMINARY REPORT

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and

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In many of the best departments, localization of the placental site by soft-tissue radiography has become an easy and reliable method.[1, 5, 6, 9, 10] With the films taken in our own less than perfect department, and interpreted by ourselves, mistakes are apt to occur, and we are embarrassed from time to time by the fallacies Chassar Moir pointed out.[9]

Attempting to find an easier method in an average department, we have lately examined patients with antepartum haemorrhage by transfemoral aortography by Seldinger's method.[7] Although our series is still small, we have been so satisfied with the results that we are anxious to bring the procedure to the attention of more of our colleagues.

Conclusions

It is said that the last thing any sensible person reads in a book is the introduction. Similarly, the first part of an article to be read is often the conclusion. To save him the trouble of turning the pages, we head our report with the conclusions, so that the reader may examine the facts that follow critically, with these deductions in mind, and draw his own inferences:

1. The safest method of placentography is by retrograde aortography—if radiation hazard is given due importance.
2. The simplest method of placentography in the average or small department is by retrograde aortography —with the help of Seldinger's apparatus.
3. The most accurate method of placentography is by retrograde aortography—in the hands of the *inexpert* interpreter.

Material

Our examination on patients with antepartum haemorrhage includes only 25 cases so far. All were women over 28 weeks pregnant, with bleeding of minimal or moderate severity, of unexplained etiology. Patients with severe haemorrhage were not included, because they are usually examined vaginally in the operating theatre by the obstetrician.

TECHNIQUE

The examination is performed under local anaesthetic (Leostesin), 45 minutes after the administration of 100 mg. of pethidine.

The patient is examined in the supine position as a rule. The apparatus devised by Seldinger[7] makes the procedure not only possible but surprisingly easy. Newcomers to our department have mastered the technique at the first attempt. We believe that Seldinger has brought aortography within reach of every radiologist who can effectively give an intravenous injection.

The technique of passing the catheter up the femoral artery into the aorta need not be fully described. One may mention again the need to puncture the skin with a pointed blade before using the trochar, to open the path for the catheter. We keep our catheters, with the ends already prepared, sterile by immersion in Zephiran, each catheter being housed separately in a straight glass-tube. When one is not prepared for an emergency, polythene catheters may be boiled for a few minutes, provided one handles them very gently while hot. All other apparatus is autoclaved and stored in a drum, ready for use.

Catheter Length. A linen measuring tape is sterilized with the rest of the apparatus, and the catheter measured before its introduction. It is easy then to determine afterwards what length of catheter lies in the artery. In most patients 9 inches of catheter introduced will reach above the level of the aortic bifurcation, yet lie well below the important branches of the aorta. The measurement dispenses with the need for a preliminary exposure of mother and child to determine the position of the catheter.

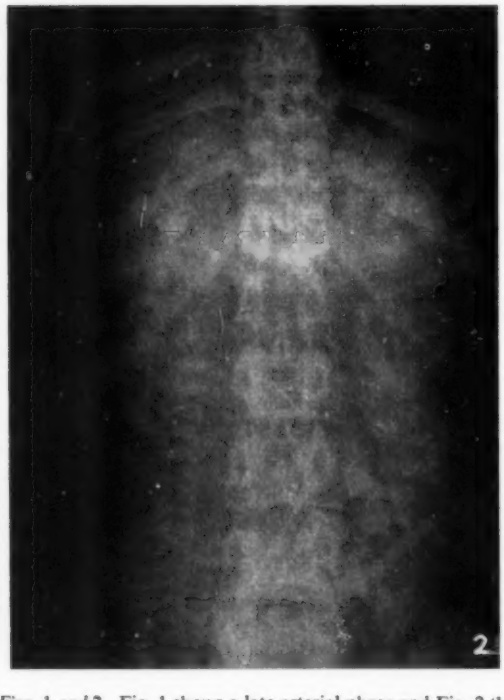
Tourniquet. Baumanometer cuffs are applied above both knees and inflated to a pressure higher than arterial pressure just before the injection is made.

Contrast Medium. Inject as rapidly as possible by hand 20-25 ml. of 50% Diaginal. Speed of injection is essential. On a few occasions mistakes in technique due to inexperience have necessitated a second, once even a third, injection without untoward effect. Sensitivity was tested by various means in different patients: Conjunctival, intravenous or intra-arterial administration.

Exposures. A single picture, taken 1½-2 seconds after completion of the injection indicates the placental site clearly in most cases. It demonstrates the rather star-like sinusoids which are characteristic of the placenta, and shows them all the more clearly because the large arteries have already emptied sufficiently not to obscure them. We have taken films at various intervals and found the contrast to persist in the sinusoids up to 10 seconds after the injection, but to fall off rapidly in density after 6 or 7 seconds. The early arterial phase, obtained by making exposures during the first second after completion of injection produces a less convincing picture of the placental site than the sinusoidal phase does.

Position.

At first all our patients were examined in the antero-posterior projection. In some of these cases critical



Figs. 1 *and* 2. Fig. 1 shows a late arterial phase and Fig. 2 the sinusoids 6 seconds later.

Fig. 3. A.P. film taken 2 seconds after completion of the injection showing typical sinusoids with placenta *en face*.

localization of a low-lying placenta situated on the anterior or posterior uterine wall was difficult. The patient was then turned on to her side and a second injection of contrast material made to obtain a lateral view. Interpretation of the lateral view is often easier.

It appears as if the placenta usually develops on the anterior or posterior rather than on the lateral uterine walls, perhaps because it has a bilateral blood supply. For this reason we now tend to prefer a single lateral view to the antero-posterior projection.

It has become our habit to refer to high and low implantation of the placenta rather than to upper and lower segments, because it is impossible to demonstrate these segments radiologically.

REACTIONS

Most patients experience a feeling of heat in the back during injection, varying in degree from a slight feeling of warmth to discomfort sufficient to make a hypersensitive patient cry out. Surprisingly enough nausea and vomiting have not occurred in any of our patients so far.

Onset of Labour.

Contrary to previous experience with 80% sodium iodide and with Diodrast,[2] uterine contractions do not appear to be stimulated by 1, 2 or even 3 injections

of 50% Diaginol. Many of our patients were at or near term, yet in no case did labour commence within the first day following the investigation.

Some but not all of our patients were given ¼ gr. of morphine on their return to the ward.

Fig. 4. Lateral film taken 2 seconds after completion of injection. This clearly demonstrates the placental site on the upper posterior uterine wall.

DISCUSSION

We have found placentography by retrograde aortic catheterization a simple procedure in an average department, and so far free from complications.

Radiation hazard is constantly in the news, and now alarming reports have reached us from England,[8] indicating a relationship between antenatal radiological examination and the development of leukaemia in children. The biggest advantage of aortography over other methods of placentography may be that the whole examination often requires only a single exposure.

The reliability of the results will still have to be assessed by a careful follow-up of a large number of cases. Already, in a series of only 25 cases, we have been mistaken in one case, in which marginal placenta praevia felt on digital examination had, at a previous arteriographical examination appeared to lie somewhat higher in the uterus. However, the error occurred at a time when we were still taking two films in each case, viz. a first exposure made during the arterial phase, which we have found to be unreliable for placental localization, and a second film taken as soon as the casette in the bucky tray could be changed by hand, which was often too late. Since we have modified our technique to that described above, the placental site has been more convincingly demonstrated.

Review of Literature

Many excellent papers have been published on soft-tissue and indirect placentography.[1, 5, 6, 9, 10] A satisfactory, non-toxic selective medium which on administration to the mother will opacify the placenta, is not yet available.[10] Meanwhile, many workers have felt that aortography might be the most reliable method of demonstrating the placental site, but have feared that it would be too dangerous for routine use. [2, 9, 10]

Translumbar aortography is certainly liable to occasional serious complications such as (1) severe bleeding from a site which cannot be controlled by pressure, (2) injection of contrast material directly into an important branch such as the renal artery, and (3) trauma to adjacent structures.[3, 4, 9] It has generally been considered necessary for translumbar aortographic placentography to be performed under general anaesthesia.[2, 9] This disadvantage in itself would preclude the method as a routine procedure in the eyes of many obstetricians.

The contrast material available in the past has been unsuitable. Weinberg[9] records the occurrence of uterine contractions as an argument against the safety of aortography during pregnancy, and Hartnett[2] states that many patients at or near term began labour immediately or shortly after the intra-aortic injection of 70% Diodrast or 80% sodium iodide.

CONCLUSION

Our small series has given us reason to believe that the objections to aortography may have been overcome, and that the hopes other workers have had for arteriographic demonstration of the placenta have not been unfounded. Perhaps larger departments than ours will be able to investigate the method more fully, modify the technique where necessary and test the reliability of the results against clinical follow-up examination.

OPSOMMING

Onder gunstige omstandighede kan die ligging van die plasenta in die meerderheid van gevalle met redelike betroubaarheid op gewone roentgenfotos van die buik bepaal word.

Aortografie kom as 'n betroubare ondersoek voor, maar tot dusver was die translumbale inspuiting van kontrasmiddels in die aorta as te gevaarlik vir roetine-gebruik beskou. Dit hou gevare in van bloeding, inspuiting in een of ander belangrike vertakking van die aorta en besering van ander naasliggende organe.

Daarteenoor staan die metode wat ons nou gebruik, om die Seldinger-buis deur die femorale slagaar in die aorta op te stoot. Die ondersoek kan sonder narkose en skynbaar sonder groot gevaar gedoen word.

Die kontrasmiddel, Diaginol, in 50% oplossing het die voordeel bo ander stowwe wat vroeër gebruik was,

dat dit nie die uterus laat saamtrek nie. In die 25 gevalle van antepartum bloeding wat ons tot dusver ondersoek het, het geboortepyne in geeneen op die eerste dag na die ondersoek begin nie.

Navorsing in England het onlangs aan die lig gebring dat roentgenbestraling gedurende swangerskap nadeliger gevolge mag hê as wat tot dusver bekend was. Aortografie het dié voordeel bo ander vorme van plasentografie dat die ondersoek gewoonlik deur 'n enkele bestraling voltooi word.

SUMMARY

The technique of placental localization by retrograde aortography is described. The results in 25 patients investigated for antepartum haemorrhage are discussed. The method is compared with some of the other procedures used, and attention is drawn to the relatively small dose of radiation received by the patient.

We owe a special debt of gratitude to the head of our department, Dr. Margaret Findlay, who has always taken a keen interest in arteriographic work and has stimulated and encouraged its development at King Edward VIII Hospital over the past few years.

Our thanks are due to Dr. S. Disler, Medical Superintendent of the King Edward VIII Hospital, for permission to publish, and to the staff of the obstetrical wards for their willing cooperation.

We wish to thank our own staff for their participation. Dr. S. Baker did some of the examinations and contributed useful suggestions.

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ADDENDUM

Since this paper was prepared, one patient gave birth to a live child on the day following aortography, and died 2 days later. Other factors were operative in this case, and the death cannot be ascribed to arteriography.

MEDICAL ASSOCIATION OF SOUTH AFRICA

MINUTES OF THE ANNUAL GENERAL MEETING

Following are the Minutes of the Annual General Meeting of the Medical Association of South Africa, held at Red Cross House, Riebeeck Street, Cape Town, on Wednesday, 3 October 1956, at 9.40 a.m.

The President (Dr. J. H. Struthers) was in the Chair, and 49 other members were present. There were 20 proxies which were declared to be in order.

1. *Notice Convening the Meeting*, published in the *Journal* of 18 August 1956, was taken as read.

2. *Minutes* of the last Annual General Meeting, held in Pretoria on 17 October 1955, were taken as read. It was proposed by Mr. Armitage, seconded by Mr. Wolfowitz and *Agreed* that they be confirmed. The Minutes were then signed by the President.

3. *Annual Report of Chairman of Federal Council:* This Report had been published in the *Journal* of 25 August 1956. The Chairman of Council (Dr. A. W. S. Sichel) moved its adoption, seconded by Dr. Goldberg. *Carried.*

4. *Financial Statement and Balance Sheet*, published in the *Journal* of 7 July 1956: It was proposed by the Honorary Treasurer (Dr. J. S. du Toit), seconded by Dr. Impey and *Agreed* that the Financial Report be adopted.

5. *Election of Auditors:* It was proposed by the Honorary Treasurer, seconded by Dr. Theron and *Resolved* that Messrs. Gurney, Notcutt & Fisher be reappointed auditors for the year 1957, at a remuneration of £200 per annum.

6. *Induction of President:* Dr. Struthers expressed his appreciation of having been allowed to serve the Association as its President during the past year, and he introduced Dr. J. S. du Toit who was to succeed him. He said he felt that there could be no more worthy recipient of the honour of being President of the Association than Dr. du Toit who had been a faithful member and worker since his return to the Union after completing his studies in Edinburgh. He had joined the Cape Western Branch of the British Medical Association as it had been known at that time, and had been Honorary Treasurer of the Branch for many years. When the Medical Association of South Africa (B.M.A.) had been formed in 1927, he had become the first Honorary Treasurer of the Association and had continued to fill that position until the present time. Dr. du Toit had been Vice-President of Congress in Cape Town in 1933 and again in 1949, and in 1952 he had received the award of the Association's Gold Medal for distinguished service to the Association and the profession. Dr. Struthers expressed the hope that Dr. du Toit would have a very happy year of office.

Dr. Struthers then installed Dr. du Toit as President with the President's insignia of office, amid acclamation.

Dr. du Toit replied, stating that he was deeply honoured at having been elected to be the Association's President after many years of service to and interest in the Association.

Dr. Sichel proposed a vote of thanks to Dr. Struthers for the conscientious way in which he had carried out his duties as President during the past twelve months. He mentioned the visit which Dr. Struthers had paid to England at the time of the Annual Meeting of the British Medical Association when he had been South Africa's representative at that meeting.

The vote of thanks was accorded with acclamation.

The meeting adjourned at 9.50 a.m.

THE ADJOURNED ANNUAL GENERAL MEETING

Following is a Report of the Adjourned Annual General Meeting of the Medical Association of south Africa held in the Jamieson Hall, University of Cape Town, Rondebosch, Cape, on 3 October 1956, at 8.15 p.m.

After the entry of the platform party, Dr. A. J. van der Merwe led the meeting in prayer.

On behalf of the City of Cape Town, the Mayor welcomed delegates attending the Federal Council meeting, saying that he hoped that their stay would be so memorable as to induce them to pay another visit. He extended to Dr. J. S. du Toit the congratulations and good wishes of himself and the City Council on his election as President of the Association, saying that he felt

that it was an honour rightly deserved after so many years of service to the Association. Cape Town, he said, was proud that one of its citizens had received this honour. Acclamation.

Dr. du Toit called on Dr. Sichel, the Chairman of Federal Council, to reply to the Mayor's address of welcome. On behalf of the Federal Council Dr. Sichel expressed the Association's thanks to the Mayor for his presence at the meeting, his welcome to the delegates and especially for his kind reference to the President. Acclamation.

A musical interlude followed, rendered by two students of the South African College of Music.

Emeritus Membership: The Secretary introduced to the President Drs. Elsie Chubb, Charles Muller and James Luckhoff, for presentation of the Association's Certificate of Emeritus Membership, adding that the Association wished also to honour Drs. J. G. Luyt and H. M. Griffiths in the same way. The latter two gentlemen had not been able to be present at the meeting.

The Certificates were presented to the recipients by the President, amid acclamation.

Bronze Medals: The Secretary read citations in respect of Dr. R. Lance Impey, Dr. J. P. de Villiers and the late Mr. L. B. Goldschmidt, whom the Association desired to honour by the award of the Association's Bronze Medal for meritorius service to the Association. The awards were presented to the first two gentlemen by the President, amid acclamation.

Silver Medal: The Secretary stated that at the meeting of the Federal Council held that day it had been decided to institute a new award known as the Association's Silver Medal, to be made for outstanding services in the field of medical research. At the meeting it had been resolved unanimously that the first recipient of the Silver Medal should be Prof. M. van den Ende of the University of Cape Town, who was a medical scientist of international repute. In due course the medal would be prepared and presented to Prof. van den Ende. Acclamation.

Hamilton-Maynard Memorial Medal: The Secretary stated that it had been agreed that two Hamilton-Maynard Memorial Medals should be awarded for the year 1955. In the circumstances it had been agreed that one should be awarded to Mr. T. Schrire for his paper entitled 'Modified Radical Gastrectomy for Cancer of the Stomach', which appeared on page 494 of the *Journal* of 2 May 1955, and the other to Dr. W. P. U. Jackson for his paper entitled 'Osteoporosis—Commonest of All Diseases'? published on page 885 of the *Journal* of 17 September 1955.

The President presented the Medal to Dr. Jackson, and in the absence of Mr. Schrire his award was received on his behalf by Prof. Jannie Louw. Acclamation.

Leipoldt Memorial Medal: The Secretary stated that the Leipoldt Memorial Meda! for 1955 had been awarded to Dr. L. Solomon, of Uppington, in recognition of his paper entitled 'Acute Dilatation of the Stomach', published on page 386 of the *Journal* of 23 April 1955. As Dr. Solomon was not present, it was agreed that the Medal should be presented to him on a suitable occasion to be arranged.

Immediate Past President: Dr. du Toit then presented to Dr. J. H. Struthers, the Immediate Past President, a minature of the President's insignia. Acclamation.

Mrs. Struthers presented to Mrs. du Toit the badge of office of President's Lady, amid acclamation. Mrs. du Toit was also presented with a bouquet of flowers.

A further musical interlude followed, after which the two lady pianists were presented with posies.

Presidential Address: Dr. du Toit then delivered his Presidential Address entitled 'Factors Concerning the Sense of Vision'. This was received with acclamation.

The meeting ended at 9.30 p.m., after which a reception was held in the Student's Union. The guests were received by the President of the Association and Mrs. du Toit, and the President of the Cape Western Branch and Mrs. Owen-Smith.

UNIVERSITEIT VAN PRETORIA

By die jaarlikse Promosieplegtigheid op Saterdag 8 Desember 1956 is die volgende grade toegeken:

FAKULTEIT VAN GENEESKUNDE

Graad van Magister in Snykunde

Hugo, Pierre André.
Muller, Lourens Hilgard.

Graad van Magister in Geneeskunde

Combrink, Johannes Michiel. (Departement Interne Geneeskunde.)
de Vaal, Pieter Steynberg. (Departement Anestesiologie.)
Hattingh, Gerhardus Hendrik Holtzhausen. (Departement Obstetrie en Ginekologie.)
Malherbe, Lodewicus Francois. (Departement Interne Geneeskunde.)
Silansky, Joseph. (Departement Radiologie.)
van den Berg, Jan Louis. (Departement Anestesiologie.)

L. J. te Groen Medalje. Vir die beste graduerende in Obstetrie en Ginekologie:

Bester, John Ernest.

Protea Holdings Pryse. Vir die beste nagraadse student in Radiologie:

Silansky, Joseph.

Vir die beste graduendes in Interne Geneeskunde:

Hill, James Michael.
Otto, Cornelius Bredenkamp.
van Heerden, Corretha.

Vir die beste graduerende in Radiologie:

Lötter, Willem Johannes.

Vir die kandidaat met die beste resultate in die finale eksamen vir die Diploma in Radiografie:

Bezuidenhout, Rachel Margaretha.

Sesjarige Baccalaureusgraad in Geneeskunde en Snykunde

Barnard, Pieter Melius.
Basson, Pieter.
Bester, John Ernest. (* Ginekologie.)
Botha, Barend Cornelius.
Burger, Pieter Zacharias.
Calitz, Frederik Jacobus Wilhelmus.
Cloete, Gert Nicholaas Petrus.
de Lange, Johanna Frederica.
du Plessis, Hennie Lodewia.
Engels, Frederik Lodewyk Christian.
Friedland, Gerald Wilfred.
Gildenhuys, Hendrik Jacobus.
Grassmann, Ruth.
Hanekom, Johannes Hendrik Lindes.
Higs, Isolde.
Hill, James Michael. (* Interne Geneeskunde.)
Kleynhans, Michael Jacobus.
Kritzinger, Carel Theodorus Möller.
Lecuona, Angus Eustace Harold.
Lötter, Willem Johannes. (* Radiologie.)
Malan, Ardon David.
Maree, Hermanus Petrus. (* Kindergeneeskunde.)
Meyer, Jacobus Herklaas.
Moolman, Maria Elizabeth.
Myburg, Carel Aäron.
Novis, Henry.
Olivier, Johannes Lodewikus.
Otto, Cornelius Bredenkamp. (* Interne Geneeskunde.)
Retief, Petrus Jacobus.
Scholtz, Trevor.
Sippel, Henry Duncan.
Swart, Hendrik Strydom.
te Groen, Frans Wilhelmus.
van den Berg, Susanna Johanna Catharina.
van Heerden, Corretha. (* Interne Geneeskunde.)
van Rooyen, Christie Marais.
van Tonder, Henning Willem Jacobus.
van Zyl, Willem Hendrik.
Venter, Pieter Jacobus.
Visagie, Hermanus Adriaan.
Von Moltke, Bernhardt Eduard von Strauss.
Vorster, Jacobus Daniel.
Vorster, Leonard. (* Radiologie.)
Vorster, Willem Frederick.

* Langs naam: Graad met lof.

* Langs vak(ke): Lof in betrokke vakke.

PASSING EVENTS : IN DIE VERBYGAAN

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering.

Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Dr. T. P. S. Mulligan, D.C.P. (London) has joined Dr. W. P. Mulligan in partnership as a Clinical Pathologist at National Mutual Buildings, 17 Church Square, Cape Town. Telephone: rooms, 2-4444 and 2-1220; residence, 7-5421.

Dr. T. P. S. Mulligan, D.C.P. (Lond.) werk nou saam met dr. W. P. Mulligan as 'n Kliniese Patoloog by National Mutual-Gebou, Kerkplein 17, Kaapstad. Telefone: spreekkamers, 2-4444 en 2-1220; woning, 7-5421.

* * *

Suid-Afrikaanse Mediese Kongres, 1957. Lede wat Durban gedurende September 1957 vir die Mediese Kongres besoek, word aangeraai om met hul naaste Suid-Afrikaanse Spoorweg Reisburo of Stasiemeester in aanraking te kom aangaande akkomodasie Dit sal korrespondensie bespaar, enige moontlikheid van dubbel bespreking verhoed en bevredigende reisreëlings verseker.

* * *

Dr. M. Jordaan, voorheen van ‚Naidous', Eytonweg, Claremont, het verhuis na ‚Rockaways', Hoofweg 501, Drieankerbaai.

REVIEWS OF BOOKS : BOEKRESENSIES

DERMATOLOGY

Dermatology. By Donald M. Pillsbury, M.A., D.Sc. (Hon.), M.D., Walter B. Shelley, M.D., Ph.D. and Albert M. Kligman, M.D., Ph.D. Pp. xix + 1331. 564 Figures. $20.00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: Section I. Applied Basic Principles in Diseases of the Skin. 1. General Considerations. 2. The Epidermis. 3. Pigment Formation. 4. Keratinization. 5. The Permeability of Skin 6. Nails. 7. Hair. 8. The Glands of the Skin. 9. The Corium and the Subcutaneous Tissue. 10. The Blood Vessels and Nerves of the Skin. 11. Basic Pathologic Patterns. 12. Fundamentals of Cutaneous Mycology. 13. Fundamental Cutaneous Bacteriology. *Section II. Basic Principles and Clinical Applications of Allergy and Hypersensitivity.* 14. Introduction—Definition and Classification of Allergic Disease. 15. Serum Sickness—Anaphylaxis, Atopy. 16. Allergy of Infection. 17. Drug Allergy. 18. Contact Dermatitis of the Allergic Type. *Section III. Principles of Diagnosis.* 19. The Medical History in Dermatologic Patients. 20. Clinical Examination and Regional Diagnosis. 21. Diagnosis: Types of Skin Lesions. 22. Distribution Patterns as an Aid to Diagnosis. *Section IV. Dermatologic Therapy.* 23. Topical and Systemic Therapy. 24. Ionizing Radiation Therapy. 25. Surgical Diathermy and Ultraviolet Light Therapy. *Section V. Cutaneous Medicine.* 26. Dermatitis and Eczema. 27. Bacterial Infections. 28. Systemic Bacterial Infections 29. Spirochetal Infections. 30. Fungal Infections. 31. Viral and Rickettsial Infections. 32. Papulosquamous Eruptions. 33. Diseases of the Blood Vessels. 34. Chronic Vesiculobullous Eruptions. 35. Acne, Acneiform Eruptions and Rosacea. 36. Diseases of the Eccrine Sweat Glands. 37. Diseases of the Apocrine Glands. 38. Disturbances in Pigmentation. 39. Drug Eruptions. 40. Disturbances in Keratinization. 41. Diseases of the Corium and Subcutaneous Fat. 42. Lupus Erythematosus, Scleroderma, Dermatomyositis, and Sarcoidosis. 43. Metabolic and Nutritional Diseases. 44. Diseases of the Hair. 45. Disorders of the Nails. 46. Dermatologic Parasitology. 47. The Lymphomas. 48. Tumors of the Skin. 49. Hereditary Cutaneous Disorders. 50. Psychocutaneous Medicine. 51. Reactions to Physical Agents. 52. Industrial Dermatoses. Index.

This is a new text-book written by members of the staff of the University of Pennsylvania. In spite of its bulk it is intended for students and general practitioners rather than for dermatologists. Let us say at once that the two former will find it admirable. A quarter of the book is devoted to the basic principles of dermatology, the rest to the diseases themselves. Descriptions are always clear and concise and written in unstilted style. The photographs are well-chosen and clearly reproduced but some humorous line drawings could well have been omitted from an otherwise dignified production.

Methods of treatment suggested are reasonable and practical details are given wherever necessary. The authors' common-sense views on radiotherapy in skin diseases will please some of my colleagues in that speciality. They state that X-ray therapy may be curative or often justified in only 3 conditions—tumours, haemangiomas and tinea capitis—and that it is occasionally indicated in chronic eczematous dermatitis, circumscribed neurodermatitis and seborrhoeic dermatitis. Some other diseases are listed under dermatoses in which X-rays are commonly used but in which their effectiveness is equivocal, and dermatoses in which X-rays are contra-indicated. If only for this chapter the book ought to be made compulsory reading for all medical students. Equally restrained (for an American text) is the section on psychocutaneous medicine.

In the preface the remark that most text-books seem to have been written to perpetuate error is quoted. The authors have succeeded reasonably well in avoiding this pitfall but it is to be hoped that plagiarists will avoid a few of their own misdemeanours. Rather more is known about para-psoriasis and cutaneous papillomatosis than they think and a study of Scandinavian and South African work would suggest that the subjects of porphyria and epidermolysis bullosa should not be in different chapters. Only key references are given, but it is obvious that the dermatological Iron Curtain, running north and south at the English Channel, proved relatively impenetrable.

These criticisms do not detract from my approval of this book as a whole, which I believe is the best of its kind published in the United States.

J.M.

BREAST FEEDING

Breast Feeding. 2nd Edition. By F. Charlotte Naish, M.A., M.D. (Cantab.) Pp. xiv + 161. 22 Illustrations. 12s. 6d. net. London: Lloyd-Luke (Medical Books) Ltd. 1956.

Contents: Preface. Foreword (by Professor A. Moncrieff). Introduction. I. The Mind of the Mother. II. The Physiology of Lactation. III. Antenatal Preparations. IV. The First Week. V. The Second Week. VI. The Danger Weeks (third to sixth). VII. Weaning. IX. Breast Trouble. X. Contra-Indications to Breast Feeding and Indications for Weaning. XI. Breast Feeding without Suckling. Index.

Most practitioners, whose doubts and fears about a sick child are often assuaged by ancillary methods—the blood in typhoid, the lumbar puncture in meningitis, the X-ray in pneumonia, still find the infant who fails to thrive a puzzling and perplexing problem. They are inclined to discard breast feeding as soon as difficulties are encountered. In this book the author sets out to enumerate these difficulties and describes ways and means of solving them. It contains instructive chapters on the physiology of lactation and antenatal supervision. Attention is drawn to the difference between the secretory activity and the propulsive action of the breast i.e. 'the automatic expulsion of the milk'.

The author singles out the first 6 weeks as the vital period during which difficulties may arise. 'The First Week' is a chatty chapter, full of common sense. The author expresses her opinion upon the time and the amount of feeding and upon the controversial question of rooming-in the mother and the baby—which in private houses, where most babies in South Africa are born, is chiefly dependent upon the parents' economic position. There is generally more rooming-in amongst the poor. Practical information is given upon the causes of failure to suck, though the statement that 'there are certain nurses who always succeed in establishing breast feeding', must refer to a unique experience. Vomiting is cursorily discussed and haemorrhage of the newborn is merely mentioned. 'The Second Week' gives useful hints on usage of the breasts, wind, vomiting, diarrhoea and constipation. Under 'third week' there is advice on refusal of the breast, 'running away of the milk', the mother's diet, diarrhoea and constipation.

A useful chapter is given on weaning. 'Contra-indications to Breast Feeding' are too wide and are a net for the unwary; they should be more explicitly pin-pointed.

The last chapter on Breast Feeding without Sucking, sums up the fundamental faith of the author, that no effort should be spared to let an infant have human milk; and it is right. Why should we lightly deprive a baby of the milk manufactured for it, of ideal composition, available at all times, and needing no preparation—and no money? This is plain common sense. But, in the reviewer's opinion, the cult of the breast must not be ruthlessly persisted in. The statement, which is repeated in this book, that breast feeding has a lower mortality and morbidity is not, in the reviewer's opinion, absolutely correct. It is subject to two qualifications: (1) Breast feeding should be coupled with regular weight-checking and, if necessary, test feeding, in order that underfeeding may be diagnosed early and corrected easily. (2) If breast feeding fails, whether owing to faults on the part of the mother or of the infant, and artificial feeding is instituted and controlled by the doctor at regular intervals, it should be as successful as breast feeding. Of course, nobody doubts the general suitability and the efficacy of the natural way of feeding; it is conducive to the comfort of the baby and to the ease of the mother, and it is psychologically satisfying. It should not be discarded by the practitioner as soon as he meets with difficulties. Nevertheless he must not try to promote breast feeding by instilling fear into the apprehensive mother. She should be told that breast milk has an ideal composition and contains antibodies and that the average mother feels better breast-feeding her infant. But the reviewer holds that she should also be told that in this scientific age dried milks are well prepared and their composition and food-value well known, and that artificial feeding, prescribed, regulated and controlled by those who have the knowledge, should not be one with less success than breast feeding. Failure calls for self-criticism on the part of the doctor.

The book includes feeding tables modified from Mary Crosse for the premature infant. A breast-milk bank is advocated. This will need an efficient organization; once achieved it may save as much life as a blood bank.

This is the second edition of an essay that was awarded the Charles Hasting Prize, which it richly deserves. Buy it and read it.

W.R.

BRITISH SURGERY

Textbook of British Surgery. Volume I: The Abdomen. Edited by Sir Henry Souttar, C.B.E., D.M. (Oxon), F.R.C.S. Pp. viii + 547. 105s. net. London: William Heinemann, Medical Books Limited. 1956.

Contents: Editor's Preface. General Preface. I. Surgical Anatomy. Abdominal Injuries. Intestinal Fistulae.—Guy Blackburn. II. Stomach and Duodenum—J. C. Goligher. III. Liver and Gall Bladder—J. M. Pullan. IV. Surgery of the Pancreas and Spleen—Rodney Smith. V. Portal Hypertension—R. E. Horton. VI. Peritoneoscopy—J. P. Hosford. VII. Acute Intestinal Obstruction—Rodney Smith. VIII. Appendix—J. H. Lees Ferguson. IX. Peritonitis—J. H. Lees Ferguson. X. Crohn's Disease—A. W. Kendall. XI. Diverticulosis of the Small Intestine—A. S. Till. XII. Neo-Natal Intestinal Obstruction—G. H. Macnab. XIII. Hernia—F. Mitchell-Heggs. XIV. The Colon—H. Lockhart-Mummery. XV. Actinomycosis—Zachary V. Cope. XVI. Rectum and Anus—Henry Thompson. Index.

The average reader presented with so imposing a title as 'Textbook of British Surgery' will expect something rather above the average in text-books and, in part at any rate, he will not be disappointed. This book (Volume I) can probably not be improved upon as a basis for preparation for the Fellowship examination, and the material is set out in a way which is exemplary for candidates, and most acceptable to examiners.

There are several chapters which are especially good—the sections on Colon and Rectum by Lockhart-Mummery and Thompson respectively, are beautifully presented. The sub-section on hydatids of the liver is more detailed than is usual in such works, and will be of interest to South African readers. The chapters on the biliary tract and pancreas are very thorough, though one might have perhaps expected more lavish illustration in support of the admirable text. The section on hernia also suffers from a scantiness of illustration where it would be most appreciated, and the section on actinomycosis hardly justifies its inclusion.

While the volume has much to recommend it, one cannot escape the feeling that it is likely to be of more use to the examination candidate than to the practising surgeon.

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SURGICAL TREATMENT OF DUODENAL ULCER

To the Editor: Your editorial[1] of 22 December 1956 needs destructive criticism or it will do harm.

Its terms 'it is said', 'it seems', 'in some cases' and 'it is generally held' are not valid where such an important step as the return to an obsolete operation is advocated.

Your whole article seems to have only the English school of surgery in mind, a school which is overly conservative to use a euphemism. Monyihan, for instance, 'introduced' (the right word) gastrojejunostomy in 1908, but only to English surgery. It had been fully tried and tested on the Continent for more than 25 years, ever since Wölfler performed the first one in 1881, and von Hacker perfected the retrocolic variation in 1885.

The statement that a case of successful treatment of a stomal ulceration by gastrectomy caused the latter to come into vogue may be true for England, but Billroth's first operation was a contemporary of the first gastrojejunostomy (1881). The Billroth II followed, curiously enough, together with von Hacker's retrocolic gastrojejunosotomy in 1885.

It may also have been that gastrojejunostomy was replaced by gastrectomy 'in the thirties' in England, but you will find no mention of the former in the late 'tens' and 'twenties' in the text-books of the Continent as a recognized treatment for uncomplicated duodenal ulcer. It had already been thrown out. The general opinion was then (to quote Bier, Braun and Kümmel), 'In general one should not do a gastrojejunostomy when there is a patent pylorus'.

As to Farquharson's two reasons for his atavistic impulses—where he states that in *some* cases of *recurrent* ulceration the acid values of the secretion of the gastric remnant were increased as much as four times—that is a very insecure pillar indeed. 'Some cases' means nothing—1%, 10%, or how many? 'Recurrent ulceration' where? In the duodenum? Unlikely. In the stomach? In that case it is an altogether different disease—not a 'recurrence'—which may occur just as well with gastrojejunostomy. Or does he possibly mean a 'stomal ulcer', for which complication jejunostomy itself is said by you to have been abandoned? At best one could, from the given facts, draw only the conclusion that patients with a high acid secretion tend to recurrences; not that gastric resection tends to cause high acid secretions.

The statement that 'if this observation be substantiated, the main purpose of gastric resection would disappear' is unjustified. One can only draw the conclusion that in 'some cases' the surgeon did not remove enough stomach. Besides, the statement is dangerous as it shows the subconscious acceptance of the fact that this theory *will* be substantiated (as is demonstrated later in your article). If this were not so, the discussion should have stopped exactly there and the results have been awaited before conclusions were drawn.

The second pillar of the argument—the 20% so-called 'failures'—it is not much better, for Farquharson's nostalgic memory must have forgotten the 'failures' of gastrojejunostomy days, as the 'statistical evidence' adduced only too clearly shows. Stomal ulcer was not the only, not even the main, cause of failure; it was only a relatively rare complication. The fact that only few cases got it does not mean that the rest were 'cured'—not by a long stretch. In those days there were dumping syndromes too, although they had no special name then—vomiting of bile, the vicious circle in which the stomach contents played an interminable merry-go-round down the afferent loop and back into the stomach again—not to forget the numerous really uncured ulcers still

bathed in irritating gastric juice, and the unknown number of cases where the gastrojejunal stoma silently closed itself. (I have had to operate on 3 such cases in the last 2 years, for recurrent duodenal haemorrhage.)

As to the relative danger of the operation in the olden days, which is now supposed to have become less—I can only quote Bier, Braun and Kümmel again: 'Gastrojejunostomy has become a well-nigh safe operation'.

Finally the statement: 'He (the surgeon) can submit the 3·5% cases that subsequently go on to stomal ulceration to partial gastrectomy, *which is usually a great success*', is rather remarkable! Do *those* cases which have two operations not suffer from the dumping syndromes and the whole gamut of ills so luridly described earlier on?

The theory you quote that 'simple gastrojejunostomy seems to be preferable to gastrectomy' depends entirely upon the forgotten 'if' mentioned higher up, and the totally forgotten other (but very real) disadvantages and sequelae of gastrojejunostomy. One has to agree with Ogilvie that it can seem only preferable to the unskilled surgeon!

Finally the inane remark by the physician, 'sympathetically' quoted by Farquharson, that 'he would run faster than the surgeon, if that surgeon wanted to remove four-fifths of his "normal" stomach'! It is because of such woolly thinking that we so often lose the right path in our work. One agrees with Farquharson that it is a sad reflection on medicine (not 'modern' medicine; the old was just as helpless) that surgery should be necessary, but he is really blaming the physician when he says this, not the surgeon.

The surgeon does not 'want to cut out four-fifths of a normal stomach to cure a small ulcer', he cuts out a sufficient part of the *abnormal* stomach to diminish the acid secretion which the physician cannot stop. If the stomach is not abnormal in its secretion, the surgeon, if he knows his work, does not cut out four-fifths of the stomach, but only sufficient to isolate the duodenum.

One has the impression that the whole idea is rooted in the emotions rather than in the 'thinking part' of the authors. It seems to be inspired by fear of the 'really big' operation, which nowadays, in competent hands, has a lesser mortality and very certainly a lesser morbidity than the nostalgically remembered gastrojejunostomy a loved old chief used to perform.

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1. Editorial (1956): S. Afr. Med. J., **30**, 1219.

ANAESTHETIC EXPLOSIONS

To the Editor: In your issue of 8 September 1956 you published an article[1] of mine under this title. Since that time a Working Party of the British Ministry of Health has published a Report on the same subject and this forms the basis of an Editorial comment in the *British Medical Journal* (issue of 24 November). The Report, based on the study of 36 explosions occurring during the administration of 15 million anaesthetics, emphasizes that 'almost always when an accident has occurred there has been some avoidable factor against which precautions could have been taken'.

My own comments were the subject of some criticism, especially as regards the estimated incidence of explosions, but I think that the British Report figures (which I give here with my own earlier estimates for comparison) show that I did not err too greatly in my estimates:

	British Report	*My Estimates*
Number of explosions per 100,000 anaesthetics (all types)	0·24	0·24
Number of explosions per 100,000 anaesthetics (inflammable agents)	0·6	2–4
Mortality rate from explosions (all types)	1 death per 5 million	1 death per 7½ million
Mortality rate from explosions (inflammable agents)	1 death per 2 million	1 death per 1 million

I erred in assuming the risk to be greater than it is when inflammable agents are used and techniques using non-explosive agents to be more commonly used than they are. The Report (which is obtainable form Her Majesty's Stationery Office, London, for 2s. 6d.) is a short, comprehensive booklet which should be in the possession of all who give anaesthetics.

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1. Jones, C. S. (1956): S. Afr. Med. J., **30**, 861.

POLIOMYELITIS RESPIRATOR CASES

To the Editor: I have read with much interest and sympathy Dr. Louis Kaplan's article[1] on respirator cases of poliomyelitis in the *Journal* of 10 November 1956. We had similar problems in Salisbury in 1954-55, but after a visit to many European respirator centres this year one has had perforce to revise many ideas concerning the best form of treatment of the life-endangering types of cases.

There is no doubt whatever that in Prof. Lassen's regimen of treatment in the Copenhagen epidemic of 1952 the introduction of treatment by intermittent positive-pressure respiration (IPPR) practically halved his former death rate. The outstanding lesson learnt is that the tank respirator can be a lethal weapon if used in the wet bulbar or bulbospinal type of poliomyelitis.

To quote a sentence from Dr. Kaplan's article: 'But it must be realized that in many of the cases it is the excessive secretion in the inaccessible smaller bronchi and bronchioles which is the basic cause of hypoventilation, pulmonary oedema, atelectasis, and finally death'. This is often the very condition that is created by or aggravated by the tank respirator, whereas he uses this as an argument against tracheotomy and IPPR. The steep Trendelenburg position in the tank respirator also has many dangers if maintained for more than a few hours.

Where there is the slightest tendency to excess mucus due to failing deglutition, and especially if there is weakness of respiration, the patient and the constantly operated and very irritating suction apparatus as wielded by the nursing attendants are both quite impotent to cope with the deadly irresistible suction of the tank respirator in drawing this mucus into the furthest recesses of the pulmonary system. Even where a tracheotomy has been performed and a suitable rubber cuff is expected to seal off the secretions above the tracheotomy, experience has shown that the tank respirator may in practice defeat this object.

On review of cases treated in the tank respirator it is strongly felt that if tracheotomy were instituted the moment frequent use of the suction apparatus becomes necessary for the removal of excess mucus in the retropharynx (which can easily retain up to 2 or 3 oz. of fluid in the recumbent position), many lives might be saved.

It is unfair to assess the effects of tracheotomy unless recourse to this operation has been taken at the earliest possible stage, still more so in any case in which treatment with the tank respirator had already commenced.

The tank respirator remains the apparatus of choice in dry cases of respiratory failure and so long as they remain dry.

In all cases adequate ventilation is the main essential and yardstick. Regular estimation of vital capacity, tidal volume and minute volume, blood pressure, CO_2-combining power of the blood, and electrolyte balance, will help one in taking adequate early steps to maintain this ventilation. For example, where atelectasis is threatened or almost established, a falling tidal volume and rising blood pressure may give warning in time for the physiotherapist to apply the assisted cough technique (*vide* article by Dr. H. Howard Stevenson, Lancet, 26 April 1956, p. 845).

On the other hand some cases will defeat our best efforts no matter what we do.

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21 December 1956

1. Kaplan, L. (1956): S. Afr. Med. J., **30**, 1073.